Behavioral Covariates of Waist-to-Hip Ratio in Rancho Bernardo

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Abstract: We examined lifestyle and dietary habits in 685 men and 943 women (mean age 67 years) who completed an interview, examination, and food frequency questionnaire in 1984–87. Waist-to-hip ratio increased with age and body mass index in both men and women. In multiple regression, waist-to-hip ratio was independently associated with smoking, alcohol consumption, and exercise in men, and with smoking and alcohol consumption in women. The data suggest that waist-to-hip ratio is affected, at least in part, by behavioral, and potentially modifiable, factors. (*Am J Public Health* 1990; 80:1358–1362.)

Introduction

Male pattern abdominal or truncal obesity, as measured by either waist-to-hip girth ratio, or ratio of subscapular to triceps skin-fold, has been implicated in the development of ischemic heart disease,[1–3] stroke,[3,4] diabetes,[5,6] hypertension,[7–9] and all-cause mortality[2] in both men and women. The association is independent of overall adiposity. In fact, in at least one study, those at highest risk for subsequent ischemic heart disease and death were the leanest subjects, with the greatest waist-to-hip girth ratios.[2]

The etiology of abdominal obesity is not clear. Studies by Bouchard[10] have shown that heredity accounts for only 20–25 percent of the variance in waist-to-hip ratio in the general population. Shimokata[11] and we[12] recently reported that cigarette smoking is related to abdominal obesity independently of age and body mass index. However, since smoking is highly correlated with other health behaviors, such as diet, alcohol consumption, and physical activity, it was unclear whether the increased abdominal adiposity seen in smokers is due to smoking per se, or is a consequence of other, associated behaviors.

In this report we have attempted to determine whether other behaviors, specifically alcohol consumption, physical activity, and diet, also contribute to the waist-to-hip ratio in a defined population.

Methods

The population for this study consisted of older adult residents of Rancho Bernardo, a geographically defined community of Caucasian upper middle-class in Southern California. These subjects were first surveyed in 1972–74 as part of the Lipid Research Clinics Prevalence Study. As previously reported,[13] the initial response rate was 82 percent. In 1984–87, 81 percent of surviving men and women participated in a follow-up evaluation by telephone and/or clinic visit. The 76 percent who were seen in clinic form the basis of these analyses.

Smoking and alcohol use were determined by standardized, self-administered questionnaires which asked subjects if they had consumed any alcohol in the past year, the frequency of their consumption, and the specific forms of alcohol consumed in an average week and in the week prior to the survey. Grams of alcohol for the week prior to the survey and for an average week were computed by means of an algorithm.[14] For smoking, participants were asked if they had ever smoked cigarettes, whether they currently smoked, the usual number of cigarettes smoked per day, and number of years smoked. Exercise was assessed by asking participants if they regularly exercised three times per week. Diet was assessed in a subsample of 737 participants with the Harvard-Willett semi-quantitative food frequency questionnaire.[15]

In clinic, a nurse measured height and weight with subjects in light clothing, without shoes. Body mass index (weight in kg/height in m^2) was calculated as an estimate of obesity. Waist and hip girths were measured over light, single thickness clothing with participants standing in an erect position with feet together. Participants were asked to bend forward and return to an erect posture. Waist girth was measured in cms at the bending point. Hip girth was measured in cms at the iliac crest. A 75 gram oral glucose tolerance test was administered in the morning after a requested 12-hour fast; diabetes was defined by World Health Organization (WHO) criteria.[16]

Among the 841 men and 1,117 women ages 50–79 who participated, 125 men and 114 women had non-insulin dependent diabetes by history or glucose tolerance test. Non-insulin dependent diabetes, which is strongly genetically determined,[17] and related to waist-to-hip-ratio was excluded from the present analyses in order to emphasize attributes which could be modified by behavior. Subjects with incomplete diabetes data (N = 84) were likewise excluded. These analyses, therefore, are based on 685 men and 943 women without diabetes. There were no other exclusions for disease, but all subjects were healthy enough to come to the clinic.

Data were analyzed using Statistical Analysis Systems (Cary, NC). Groups were compared using analysis of variance; age and age and body mass index-adjusted values for waist-to-hip ratio were compared by analysis of covariance. Pearson correlation coefficients were used to assess the correlation of waist-to-hip ratio with the dietary variables. Waist-to-hip ratio was sufficiently normally distributed not to require transformation. Multiple linear regression was used to determine the independent contributions of the behavioral variables to waist-to-hip ratio. All probabilities reported are for two-tailed tests.

Results

The mean age of both men and women was 67 years (Table 1). Men were heavier and had a higher waist-to-hip ratio than women. BMI decreased with age in men ($r = -.19$, $p < .001$) but not in women. Waist-to-hip ratio correlated with BMI in both men and women, ($r = .40$; $r = .31$, respectively).

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TABLE 1—Clinical and Behavioral Characteristics of Men and Women Ages 50–79 in Rancho Bernardo

	Men N = 685	Women N = 943
Age (mean years)	67.5 (8.3)	67.6 (8.2)
BMI (mean kg/m²)	26.2 (3.3)	24.4 (3.7)
Waist-to-Hip Ratio	0.915 (.050)	0.789 (.062)
Smokers		
% Never	28	47
% Former	60	37
% Current	12	16
Cigarettes per day* (mean)		
Former	26 (16)	16 (15)
Current	24 (17)	16 (13)
% Drinkers	75	65
Alcohol past week** (mean gms)	142 (126)	102 (92)
Alcohol average week** (mean gms)	152 (123)	111 (88)
% Exercisers	87	80

Values are mean ± SD.
*among smokers
**among drinkers

When adjusted for BMI, waist-to-hip ratio increased with age in both men and women (r = .11, r = .23, respectively).

Almost two-thirds of the men and over one-third of the women had smoked in the past, but only 12 percent of the men and 16 percent of the women were current smokers. Over two-thirds of men and women reported drinking at least one or two drinks per month and most reported regular physical activity. Among users, men smoked more cigarettes and drank more alcohol than women.

Waist-to-hip ratio was higher in men and women who had ever smoked compared to those who had never smoked, and was higher in the upper quartile of drinkers (>9 oz/wk for men; >7 oz/wk for women) compared to all others. Waist-to-hip ratio was lower in men and women who reported regular exercise compared to those who did not (Table 2).

Waist-to-hip ratio, adjusted for age and BMI, was lowest in never smokers, intermediate in former smokers, and highest in current heavy smokers (Figure 1). The magnitude of the difference in waist-to-hip ratio between never smokers and current heavy smokers was as large in the women as in the men, despite the fact that within each category the women smoked fewer cigarettes per day.

Figure 2 shows mean waist-to-hip ratios by five drinking categories based on alcohol consumption in the week prior to the clinic visit week. Similar results (not shown) were found based on alcohol consumption in an average week. In both men and women, the heaviest drinkers had the highest waist-to-hip ratios.

Correlations between a number of dietary variables from the Harvard-Willett food-frequency questionnaire and waist-to-hip ratio were examined in 335 men and 402 women, shown in Table 3. Among the dietary variables, percent calories from alcohol was positively correlated with waist-to-hip ratio in men and women, as was absolute alcohol intake (data not shown). The only other dietary variable significantly correlated with waist-to-hip ratio was percent calories from carbohydrate (exclusive of alcohol) which was inversely correlated with waist-to-hip ratio in men.

Cigarettes per day for former and current smokers, alcohol as a continuous variable, and exercise were entered into a multiple linear regression model with age and BMI. There was no significant interaction of smoking with alcohol intake in the model. As shown in Table 4, in both sexes, age, BMI, alcohol intake, and smoking were independently associated with waist-to-hip ratio. Exercise was independently associated with waist-to-hip ratio in men but not in women. Overall, these attributes explained 21 percent of the variance in waist-to-hip ratio in men and 16 percent in women.

Discussion

The study confirms observations by Shimokata,[11] and extends earlier observations in this population[12] by demonstrating that effects of smoking and drinking on waist-to-hip ratio are independent and additive.

The fact that the increase in waist-to-hip ratio associated with drinking was accounted for by the heaviest drinkers is consistent with one possible mechanism, an increase in adrenal cortisol production which has been reported in alcoholics and has been termed "alcohol-induced pseudo-Cushing's syndrome."[18,19] It is well-known that central obesity is a prominent feature of Cushing's syndrome.[20] Although pseudo-Cushing's is apparently a relatively uncommon complication of alcoholism,[21] it is possible that a lesser degree of abnormality in cortisol secretion associated with heavy drinking could affect waist-to-hip ratio in the absence of clinical Cushing's syndrome.

The association of smoking with increased waist-to-hip ratio in women may be explained by the fact that women smokers have increased levels of adrenal androgens[22,23]

TABLE 2—Mean Waist-to-Hip Ratio by Presence or Absence Behavioral Variables

	Men			Women		
	N = 685			N = 943		
Behavior	Presence	Absence	Difference (95% CI)	Presence	Absence	Difference (95% CI)
Smoker (ever)						
Unadjusted	.918	.906	.012 (.004, .020)	.794	.784	.010 (.002, .018)
Age adjusted	.918	.906	.012 (.004, .020)	.794	.784	.010 (.003, .018)
Age and BMI adjusted	.918	.906	.012 (.004, .019)	.795	.783	.012 (.004, .019)
Drinker (upper quartile vs all others)						
Unadjusted	.928	.912	.016 (.007, .026)	.804	.786	.018 (.007, .028)
Age adjusted	.928	.912	.016 (.007, .026)	.803	.787	.016 (.006, .026)
Age and BMI adjusted	.929	.912	.017 (.008, .026)	.803	.787	.016 (.007, .026)
Exerciser (regular)						
Unadjusted	.912	.933	−.021 (−.010, −.032)	.787	.797	−.010 (−.001, −.020)
Age adjusted	.912	.934	−.022 (−.010, −.033)	.787	.797	−.010 (−.001, −.020)
Age and BMI adjusted	.913	.930	−.017 (−.007, −.027)	.789	.797	−.004 (.005, −.013)

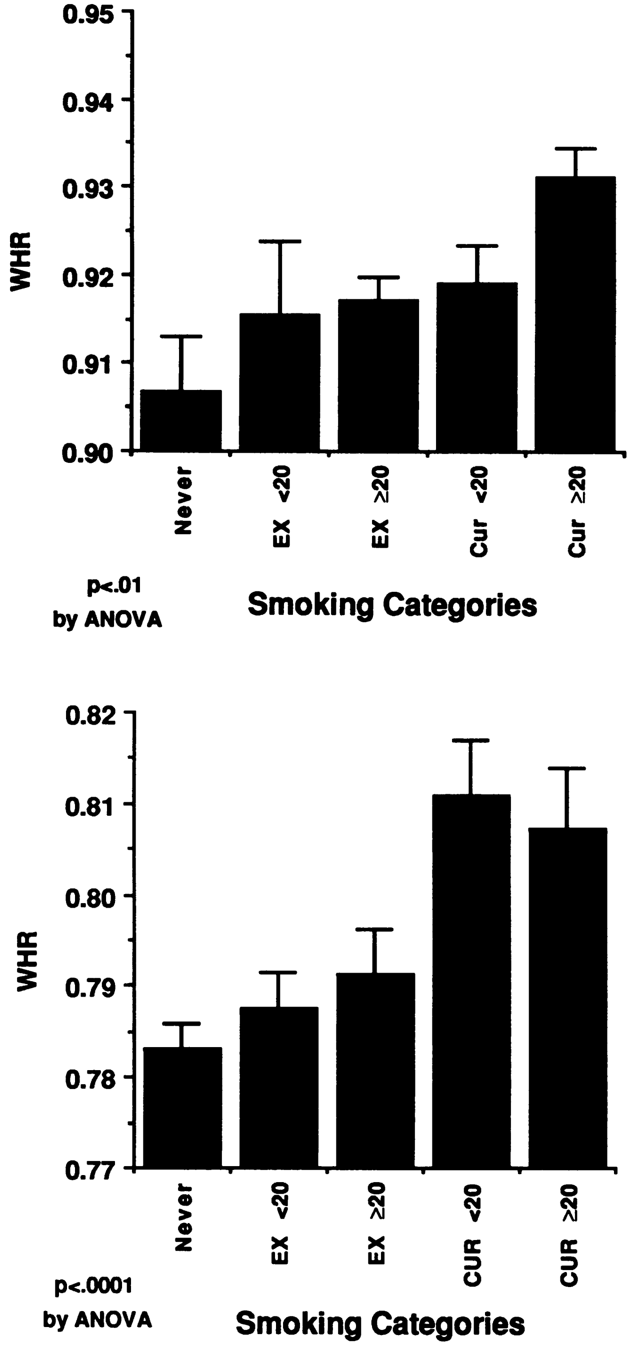

FIGURE 1—Mean Waist-to-hip Ratios Adjusted for Age and BMI by Category of Smoking: Never Smokers, Exsmokers of <20 Cigarettes per Day, Exsmokers of ≥20 Cigarettes per Day, Current Smokers of <20 and ≥20 Cigarettes per Day (Men are shown in the upper panel; women in the lower.)

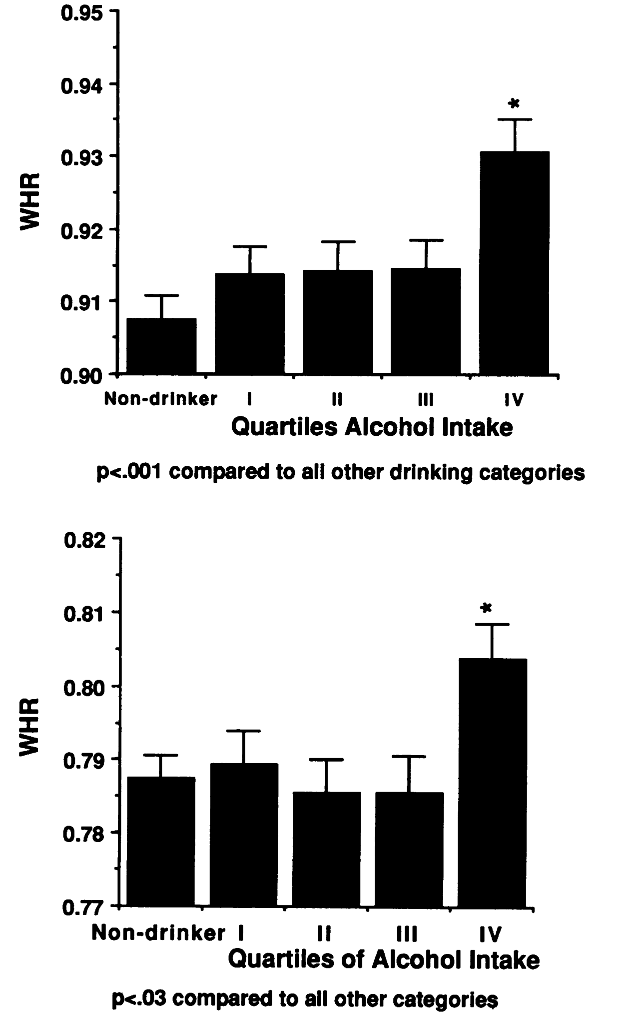

FIGURE 2—Mean Waist-to-Hip Ratios Adjusted for Age and BMI by Drinking Categories: Non-drinkers and Four Quartiles of Drinkers
(Men are shown in the upper panel; cutpoints for the quartiles are as follows: I < 2 oz/wk, II 2–4.6 oz/wk, III 4.6–9 oz/wk, IV > 9 oz/wk. Women are shown in the lower panel; cutpoints for the quartiles are as follows: I < 1 oz/wk, II 1–3.3 oz/wk, III 3.3–7 oz/wk, IV > 7 oz/wk.)

which have been shown to be associated with upper body obesity.[24] Increased cortisol levels in postmenopausal women smokers[23] also may contribute to increased abdominal obesity. In men, smoking is associated with increased adrenal androgens and estrogen, however, there are few data on the association of sex hormones with waist-to-hip ratio in men.[25] One study showed the only difference between men with higher vs lower waist-to-hip ratio was a lower sex hormone binding globulin in the former group.[26] In the data presented here, the association of smoking with waist-to-hip ratio was stronger in women than men, consistent with an effect of increased adrenal androgens. Adrenal androgens may have a less obvious effect in men, who have high levels of testicular androgens.

Except for alcohol, there appeared to be little relationship between dietary factors and waist-to-hip ratio other than the inverse relationship with carbohydrates in men. This association could be a chance finding, reflecting the multiple diet comparisons. Alternatively, a high carbohydrate diet may be associated with a healthy lifestyle and exercise.

The apparent sex difference in the relationship of exercise to waist-to-hip ratio is compatible with metabolic studies which show different effects of exercise on fat mass in men compared to women.[27] After a physical training program, obese men show a decrease in total body fat, whereas women do not.[28–30] Since waist-to-hip ratio is highly correlated with percent body fat,[31] exercise could have a greater effect on waist-to-hip ratio in men than in women.

These findings are based on older, White men and women, and may not be generalizable to younger sub-

TABLE 3—Correlations of Waist-to-Hip Ratio with Dietary Variables as Measured by the Harvard-Willett Food Frequency Questionnaire

	Men N = 335	Women N = 402
Total calories		
Unadjusted	.04	.02
Age and BMI Adjusted	.10	.01
% Calories from Alcohol		
Unadjusted	.19§	.15#
Age and BMI Adjusted	.25**	.18†
% Calories from Carbohydrate		
Unadjusted	−.25**	−.06
Age and BMI Adjusted	−.25**	−.03
% Calories from Fat		
Unadjusted	.04	−.08
Age and BMI Adjusted	.01	−.11
% Calories from Protein		
Unadjusted	.08	−.02
Age and BMI Adjusted	.00	−.03
Fiber, dietary		
Unadjusted	−.14*	−.08
Age and BMI Adjusted	−.09	−.00
Caffeine		
Unadjusted	.09	−.05
Age and BMI Adjusted	.07	−.01
Sucrose		
Unadjusted	−.06	−.06
Age and BMI Adjusted	−.05	−.04

*p = .0114, #p = .0023, †p = .0018, §p = .0005, **p = .0001

TABLE 4—Multiple Linear Regression of Behavioral Variables on Waist-to-Hip Ratio

	Men*		Women**	
	β	Standard Error	β	Standard Error
Age (years)	.00066	0.0002	.00173	0.0002
BMI (kg/m^2)	.00618	0.0005	.00537	0.0005
Alcohol (mean gms/past week)	.00005	0.00001	.00006	0.00002
Cigarettes (mean no.)	.00027	0.0001	.00034	0.0001
Exercise (Y/N)	−.01520	0.0052	−.00288	0.0046

*R^2 = 0.21
**R^2 = 0.16

jects or to minorities. However, the age group studied is that in which "middle-aged spread" is classically manifest. The analyses were based on self-reported behaviors, and it is possible that these were inaccurately reported by the participants. Probable validity of the reported alcohol intake is strengthened by concordance with aspartate aminotransferase activity and HDL-cholesterol in this cohort.[32] Likewise, the validity of self-reported exercise in this cohort is strongly suggested by its concordance with pulse and HDL-cholesterol.[33] In any event, misclassification of self-reported behaviors usually serves to obscure an association, not create one.[34]

The fact that waist-to-hip ratio appears to be determined, at least in part, by behavioral factors has implications for intervention. This risk factor for increased morbidity and mortality may be modifiable by interventions aimed at changing the behaviors which affect it. This would be expected to have a favorable impact on general, and specifically, cardiovascular health.

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REFERENCES

1. Donahue RP, Abbott RD, Bloom E, Reed DM, Yano K: Central obesity and coronary heart disease in men. Lancet 1987;2:821–824.
2. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G: Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. Br Med J 1984;288:1401–1404.
3. Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjostrom L: Distribution of adipose tissue and risk of cardiovascular disease and death: a 12-year follow up of participants in the population study of women in Gothenburg, Sweden. Br Med J 1984;289:1257–1261.
4. Welin L, Svardsudd K, Wilhelmsen L, Larsson B, Tibblin G: Analysis of risk factors for stroke in a cohort of men born in 1913. N Engl J Med 1987;317:521–526.
5. Ohlson LO, Larsson B, Svardsudd K, Welin L, Eriksson H, Wilhelmsen L, Bjorntorp P, Tibblin G: The influence of body fat distribution on the incidence of diabetes mellitus: 13.5 years of follow-up of the participants in the study of men born in 1913. Diabetes 1985;34:1055–1058.
6. Haffner SM, Stern MP, Hazuda HP, Pugh J, Patterson JK: Do upper-body and centralized adiposity measure different aspects of regional body-fat distribution? Relationship to non-insulin-dependent diabetes mellitus, lipids and lipoproteins. Diabetes 1987;36:43–51.
7. Blair D, Habicht JP, Sims EA, Sylvester D, Abraham S: Evidence for an increased risk for hypertension with centrally located body fat and the effect of race and sex on this risk. Am J Epidemiol 1984;119:526–540.
8. Weinsier RL, Norris DJ, Birch R, *et al:* The relative contribution of body fat and fat pattern to blood pressure level. Hypertension 1985;7:578–585.
9. Selby JV, Friedman GD, Quesenberry CP: Precursors of essential hypertension: The role of body fat distribution pattern. Am J Epidemiol 1989;129:43–53.
10. Bouchard C: Genetic factors in the regulation of adipose tissue distribution. Acta Med Scand 1988:Suppl 723:135–141.
11. Shimokata H, Muller DC, Andres R: Studies in the distribution of body fat. III. Effects of cigarette smoking. JAMA 1989;261:1169–1173.
12. Barrett-Connor E, Khaw KT: Cigarette smoking and increased central adiposity. Ann Intern Med 1989;111:783–787.
13. Criqui M, Barrett-Connor E, Austin M: Differences between respondents and non-respondents in a population-based cardiovascular disease study. Am J Epidemiol 1978;108:367–372.
14. Criqui M, Cowan LD, Tyroler HA, Bangdiwala S, Heiss G, Wallace RB, Cohn R: Lipoproteins as mediators for the effects of alcohol consumption and cigarette smoking on cardiovascular mortality: results from the Lipid Research Clinics Follow-up study. Am J Epidemiol 1987;126:629–637.
15. Willett WC, Sampson L, Stampfer MJ, *et al:* Reproducibility and validity of a semiquantitative food frequency questionnaire. Am J Epidemiol 1985;122:51–65.
16. Diabetes mellitus: report of a WHO study group. World Health Organization Technical Report Series 727. Geneva: WHO 1985.
17. Barnett AH, Eff C, Leslie RDG: Diabetes in identical twins: a study of 200 pairs. Diabetologia 1981;20:87–93.
18. Swartz CM, Dunner FJ: Dexamethasone suppression testing of alcoholics. Arch Gen Psychiatry 1982;39:1309–1312.
19. Kapcala LP: Alcohol-induced pseudo-Cushing's syndrome mimicking Cushing's disease in a patient with an adrenal mass. Am J Med 1987;82:849–856.
20. Hiramatsu R, Yoshida K, Stao T: A body measurement to evaluate the pattern of fat distribution in central obesity: A screening and monitoring technique for Cushing's syndrome. JAMA 1983;250:3174–3178.
21. Kirkman S, Nelson DH: Alcohol-induced pseudo-Cushing's disease: A study of prevalence with review of the literature. Metabolism 1988;37:390–394.
22. Khaw K-T, Tazuke S, Barrett-Connor E: Cigarette smoking and levels of adrenal androgens in postmenopausal women. N Engl J Med 1988;318: 1705–1709.
23. Friedman AJ, Ravnikar VA, Barbieri RL: Serum steroid hormone profiles in postmenopausal smokers and nonsmokers. Fertil Steril 1987;47:398–401.
24. Kissebah AH, Evans DJ, Peiris A, Wilson CR: Endocrine characteristics in regional obesities: role of sex steroids. *In:* Vague J, *et al.*, (eds): Metabolic Complications of Human Obesities. Amsterdam: Elsevier Science Publishers B.V., 1985.
25. Barrett-Connor E, Khaw K-T: Cigarette smoking and increased endogenous estrogen levels in men. Am J Epidemiol 1987;126:187–192.
26. Stefanick JL, Williams PT, Krauss RM, Terry RB, Vranizan KM, Wood

PD: Relationships of plasma estradiol, testosterone and sex hormone-binding globulin with lipoproteins, alolipoproteins and high density lipoprotein subfractions in men. J Clin Endocrinol Metab 1987;64:723–729.
27. Bjorntorp PA: Sex differences in the regulation of energy balance with exercise. Am J Clin Nutr 1989;49:958–961.
28. Despres JP, Bouchard C, Savard R, Tremblay A, Marcotte M, Theriault G: The effect of a 20-week endurance training program on adipose-tissue morphology and lipolysis in men and women. Metabolism 1984;33:235–239.
29. Krotkiewshi M, Bjorntorp P: Muscle tissue in obesity with different distribution of adipose tissue. Effects of physical training. Intl J Obesity 1986;10:331–341.
30. Tremblay A, Despres JP, Leblanc C, Bouchard C: Sex dimorphism in fat loss in response to exercise-training. J Obesity Weight Reg 1984;3:193–203.
31. Terry RB, Wood PD, Haskell WL, Stefanick ML, Krauss RM: Regional adiposity patterns in relation to lipids, lipoprotein cholesterol and lipoprotein subfraction mass in men. J Clin Endocrinol Metab 1989;68:191–199.
32. Barrett-Connor E, Suarez L: A community study of alcohol and other factors associated with the distribution of high density lipoprotein cholesterol in older vs younger men. Am J Epidemiol 1982;115:888–893.
33. Reaven PD, McPhillips JB, Barrett-Connor EL, Criqui MH: Leisure time exercise and lipids and lipoproteins in an older population. (Abstract) J Am Geriatr Soc 1990;38 (in press).
34. Beaton GH, Milner J, *et al:* Sources of variance in 24-hour dietary recall data: implications for nutrition study design and interpretation. Am J Clin Nutr 1979;32:2456–2559.